Death moment estimation in stillbirth

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Abstract: The stillbirth represents an important number, yet, of the death among new born. The current technologies cannot eliminate such cases from the statistics. Nevertheless, the technology allows deep investigations that help on more precise results on finding out the moment on fetus death. The death cause is on big forensics interest as well, from the mechanism point of view. The intrauterine fetal maceration is an essential element to establish the death moment. There are reported studies based on this parameter. Some morphology details, correlated with macro and microscopic information defined some death time ranges.

Key Words: intrauterine fetal death, fetal maceration, death time.

INTRODUCTION

Despite current technologies that allow deeper and better investigations and interventions, and even if the reported cases number decreased since 2000, the fetal death still matters on the statistics among infants and new born. For instance, in USA, in 2009, the incidence was of 1 case of every 160 births [1].

In 2005 there were reported around 2.6 million of stillbirth, meaning around 7178 cases per day. Most of them were reported on the developing countries [2]. 98% were reported in low or average income countries. About half of cases were reported as associated to the intrapartum moment that has the biggest risk, between 10% in developed countries to 50% in the South of Asia. In 2007, ACOG mentioned on a statistic that about 50% on the perinatal deaths are intrauterine fetal deaths. The Centre for Disease Control and Prevention classifies the fetal intrauterine death in 3 types, such as early (between 20 to 27 weeks of pregnancy), late (between 28 and 36 weeks) and on term (over 37 weeks). Statistics of this institution says that 1% of all pregnancies meet this result, every year happening around 24000 stillbirths in USA. The same percent includes death infants during the first year of life.

Between 1980-1991, in the Brigham and Women’s Hospital, 150 cases of stillbirths were reported. For every case, the moment of death was precisely determined. The cardiac activity was monitored, the antenatal fetal cardiac beats were tested, fetal echographia was run and, in general, continuous careful monitoring was performed. All fetal autopsies were developed using the protocol, the fetuses kept at 5 degrees Celsius until the autopsy moment, the fetal tissues were fixed in formaldehyde 10%, the blocks were de-waxed and the blades where fixed with coloration HE [9-11].

There are various definitions of fetal death. One says that the fetal death is the conception product with the weight over 500g, the age at least 22 weeks and the length of at least 25 cm. Another one includes a stillborn fetus without vital signs, the age over 24 weeks, the weight at least 500g. The American Congress of Obstetrics and Gynecologists defines the fetal death that happens during the pregnancy at any age over 20 weeks [3]. WHO recommends as definition the fetal death that occurs when a fetus is born with no vital signs, at or over 28 weeks of pregnancy age [2].

A prominent issue on fetal death is the risk of
associated complications the mother carrying a death fetus might meet. From the forensic point of view, the interest is focused on both the death moment and the cause of death, by the nature of the place it happens.

**Causes**

The endless investigation possibilities currently available, allowed wider scaling of the factors that produce stillbirths. The incidence still at high values is catalysing the scientific interest on establishing the medical, legal and moral milestones on such cases. Either maternal or fetal causes might be identified, either obstetrics or placentas conditions. Even if the intrauterine death was reported on various race, ethnicity and income conditions, no matter the mother age, still there are women likely to meet such an event.

Maternal causes might include the obesity as the most prevalent risk factor (20%) that doubles the stillbirth statistics. The association with the smoke or arterial hypertension is increasing it even more. The race membership (the Hispanics, Asians, native Americans and non-Hispanics report under 6 cases at every 1000 births while the black non-Hispanic population 11.25 for every 1000), the very young or over 35 years old (congenital death, chromosomal abnormalities) add more statistics on the stillbirth, together with the independent marital status. The maternal causes would include previous multiple births, some preexistent medical conditions. In 10% was established as stillbirth cause the prevalence of some diseases such as the diabetes type I or II that increases the risk with about 2,5 (about 17% of the cases), producing intrauterine hypoxia, congenital abnormalities etc., the arterial hypertension that caused placenta brake etc. The thyroid, kidney, asthma, systemic erythematous lupus, the APL syndrome, and the thrombophilia add more stillbirth risks on the balance too. Pregnancy loss, vascular thrombosis, inflammations, thrombose, mutations, placentae insufficiency come up. The drug addiction and the smoking affect the fetus growth, hypoxia. The cocaine was reported and placenta break cause. The alcohol is a very important factor as well, the heroine withdrawal and the exposure to various dangerous substances (pesticide) and radiations too. The mother infections such as the pneumonia, the appendicitis, pyelonephritis, the infection with influenza virus, the systemic infections (the intraamniotic infection generates early labour and eventually postpartum death), the intrauterine asphyxia – acute (prolapse, compression, birth associated trauma), subacute (infections, fetal cord failure) or chronic (placenta pathologies), post-maturity (gestation over 42 weeks leads to P insufficiency and perinatal hypoxia, stillbirth) where identified as maternal causes [1].

The fetal causes, reported on about 25% of the cases, includes three types of stillbirths, based on the birth moment, meaning between 24-27 weeks, between 28-36 weeks and over 37 weeks of pregnancy (4). Between 24-27 weeks, the fetal causes are the infections (19%), based on the various abnormalities (14%) and abortion (14%), between 28-36 weeks based on unexplained conditions (26%), IUGR (19%) and abortions (18%), and over the 37th week the causes are not known (40%), IUGR (14%) and because of the abortions (12%) [4]. The fetal causes might be split on:

- **genetic abnormalities** – the increased incidence was reported especially on the second quarter of the pregnancy. The frequent abnormalities include X monosomy (23%), the trisomy 21 (3%), the trisomy 18 (21%), the trisomy 13 (8%). During autopsy in the stillbirth cases, there were reported around 25% of cases with unique malformations (40%), multiple malformations (40%) and dysplasia (20%), and 75% of cases with genetic abnormalities, hard to identify by current cytogenetic analyses (microdeletions cr). There were reported deadly abnormalities at the male fetuses and cases where the hereditary thrombophilia was associated with stillbirth;

- **infections** – the action mechanism supposes infections or placenta harms, often associated with severe maternal illness. The infection's causes might include microorganisms, especially E choli, streptococcus B, urea plasma urealyticum in developed countries or syphilis, malaria, toxoplasma Gondi, leptospirosis, listeria mono, Q fever, Lime illness in underdeveloped countries. In the viral infection cases, it was isolated the B-19 parvovirus (over 8% of stillbirth). In such cases the virus goes through the placenta, infects the fetal erythropoietic tissue, leads to severe anemia and/or myocardia conditions. The enteroviruses and echoviruses lead to placenta inflammations, myocarditis, hydrops. The infection with cytomegalovirus leads to placenta conditions in about 2% of pregnancies. In half cases the medical condition is inherited from the mother to the fetus;

- **major malformations** – 4-26% of the stillbirth cases. They include cases of neural tube incomplete closure, abdominal wall defects, problems with the gastrointestinal tract, the premature closure of FO, right cord hypoplasia, unique umbilical arteria etc.;

- **IUGR (intrauterine growth restriction)** – is reported in about 7-15% of stillbirths. The common causes of this condition include arterial hypertension induced by the pregnancy, the thrombophilia, the drug and alcohol abuse, malnutrition, placenta abnormalities etc. The worst complication is the in utero fetal death.

Other conditions associated to the fetal death might include Rg incompatibility with erythrocytes alloimmunization, uterus malformations, maternal trauma in domestic violence, auto accidents or other situations with mechanical compression, obstetric conditions such as the cervix insufficiency, premature labour, pre-eclampsia.
Diagnostic

The recommended investigations in stillbirth cases are complex and include on one side the establishing of the maternal and family medical history and on the other side echographies, blood tests, toxicology, maternal microbiological tests, before the birth. The diagnosis is established with no doubt, by fetal echography. The fetal hearth pulsations are confirmed this way. Most often is very difficult to establish the precise death cause, either when complex evaluations are run. Such unexplainable phenomena are common to the third quarter of pregnancy. After the birth external fetus examination must be done, fetal blood tests, microbiological tests, histopathological tests on the placenta, the membrane and umbilical, cytogenetic tests, imagistic tests and eventually the autopsy on the fetus. The fetal tissue of a stillbirth is suffering specific changes, such as nuclear basophilic loss on the fixed tissues, progressive cytoplasmic eosinophilia and the loss of the main cellular details [5].

The histological criteria for the estimation of the precise moment of death include the nucleonic basophilic coloration loss in more than 1% of the cells of the organ specific regions, the total loss of the nucleonic basophilic coloration on the whole organ (100% of the cells), the tracheas cartilaginous matic basophilic coloration loss, the epithelial mucus detach from the bronchia, gastrointestinal tract or uterus.

From the forensic point of view, the precise death moment is of interest. The external examination is one very important source of information and estimation. The main parameter is the maceration level of the skin, together with the general status of the fetus. The maceration supposes the colour loss and the visceral fluidization and fluids accumulation in the body cavities. The maceration level in the fetal death is an indicator of the range between the death moment and the labour time. It is influenced by the maternal fever, placenta infections, the amniotic liquid volume, the fetal hydrops presence, the fetal prematurity, the time since the membrane breach. The changes produced by the maceration in the intrauterine fetal detach, based on the death moment, might be structured as following:

- <6h – skin face hyperemia and petechial hemorrhages on chest;
- >6h – skin areas desquamation, the size over 1 cm. Vesicular and C.O. discoloration are present;
- 12h – skin face, posterior thorax and abdomen desquamation;
- 18h – desquamation of over 5% of the body surface or desquamated areas of the body, on at least 2 locations of the body;
- 24h – brown skin abdomen and bulging appearance and moderate desquamation;
- 36h – skull compression and skin sliding;
- 48h – desquamation on more than 50% of the body surface;
- 72h – desquamation on more than 75% of the body surface;
- 96h – overlapping of the cranial sutures;
- 1 week – brown coloration and massive scratching, open mouth and collapse of the cap and laxity/dislocation of sutures;
- 2 weeks – partial or total mummification (dehydration, compression, brown colouring)[9].

The first signs of the fetal maceration are those of the skin, in the form of a feeling of slippery skin, about 6 hours, certainly 12 hours after the intrauterine death. The epidermis can be easily removed upon oblique pressure dermis, exposing a red, bright, wet surface. Approximately 48 hours after death, internal fetal organs and tissues show purple discoulouration due to hemolysis and red cell number decrease. Colored, dark fluids accumulate in the serous cavities. At 4, 5 days, the bones of the head are separated from the dura and the periost.

In the first 2, 3 days after death, the fetus has a sanguineous appearance, at 3-8 days visceral hepatic and cerebral changes appear. At 8-12 days, the discoulouration of the epidermis, bloating of the bones including skull bones, occur. At 15-30 days the fetus is turned into a gelatinous mass.

CASE PRESENTATION

This case is that of a dead, female newborn, 39 weeks old.

Pregnancy evolution: the mother was monitored from the 6th week of pregnancy, when she also was diagnosed with vaginitis, she followed monthly checks, she had at the 10th week of pregnancy, imminence of abortion, secondary anemia, mycotic vaginitis and pharyngotracheytis for which Augmentin 625X and anti-thermics were recommended, at the 24th and the 30 weeks she had imminent abortion, at the 34th week of pregnancy she was obese, weighing 85kg, height 150 cm, blood pressure 110/60 mm Hg, modified basal glycaemia, vaginitis, anemia. Medical control for diabetic and nutritional diseases was recommended, with 7 days before delivery was established the diagnosys of 38-39 weeks pregnancy, FVU, cranial presentation, gestational diabetes, gestational edema, gestational arterial hypertension. The ultrasound investigation revealed a 38-week and a 4-day pregnancy age, in evolution, MCF+, MAF+. The patient refused cesarean surgery, expressing the wish to deliver naturally, then was admitted with diagnosis of “IGIP, 39 weeks pregnancy, FVU, cranial MI presentation, CUD, gestational diabetes”, non - systemic MI - CUD, A HC - the mother of the patient shows non-insulin dependent diabetes mellitus, the clinical examination indicates apparent normal status, the local examination indicates pregnancy-specific changes, blood glucose 129.5.
Table 1. The labor progression

<table>
<thead>
<tr>
<th>Time</th>
<th>Col, expansions, membrane</th>
<th>Amniotic fluid aspect</th>
<th>Frequency BCF/min</th>
<th>Contraction range (at 5min)</th>
<th>Contraction - tonus</th>
<th>Pain, behavior, medication, analgesics</th>
</tr>
</thead>
<tbody>
<tr>
<td>23:45</td>
<td>M 2-3 cm</td>
<td>MR sp LA clear</td>
<td>120-132</td>
<td>40&quot;</td>
<td>N</td>
<td>Tas 60 mmHg AV 100/50/min</td>
</tr>
<tr>
<td>00:45</td>
<td>M 2-3 cm</td>
<td>MR sp LA clear</td>
<td>124</td>
<td>45&quot;</td>
<td>N</td>
<td>TA 115/70 mmHg AV 120/70/min</td>
</tr>
<tr>
<td>01:45</td>
<td>M 3-4 cm</td>
<td>MR sp LA clear</td>
<td>120</td>
<td>50&quot;</td>
<td>N</td>
<td>TA 110/60 mmHg AV 90-100/min</td>
</tr>
<tr>
<td>02:45</td>
<td>Ap 3-4 cm</td>
<td>MR sp LA clear</td>
<td>124</td>
<td>60&quot;</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>03:45</td>
<td>Ap 3-4 cm</td>
<td>MR sp LA clear</td>
<td>110-132</td>
<td>60&quot;</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>04:45</td>
<td>Ap 4-5 cm</td>
<td>MR sp LA clear</td>
<td>120-140</td>
<td>60&quot;</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>06:00</td>
<td>Ap 6-7 cm</td>
<td>MR sp LA clear</td>
<td>80-90</td>
<td>60&quot;</td>
<td>N</td>
<td></td>
</tr>
</tbody>
</table>

Given the labour progression (see the Table 1), it was decided to extract the fetus by caesarean intervention. A female fetal cranial presentation of 4660 g was performed. The presence of exitus, modified skin, umbilical cord edema, thick, hematic.

**External examination:** newborn female, 52 cm waist, 4570 g weight, elongated cephalic end, 34 cm skull perimeter, normal implanted ears, 1.6 cm long hair, red, glossy skin with detachment of derma - facial, cervical, bilateral arms and forearms, thorax anterior and partial abdominal, torsional lateral, posterior, abdominal lateral, lumbar and entire buttocks, partial anterior right anterior, thighs and shanks total bilateral posterior vernix caseosa cervical anterior to the insertion of bilateral dorsal ear and inguinal palm, 40 cm trunk perimeter, hand toes overlapping fingers, toenails to the same level with the fingers, the large labia cover small labia, seemingly traumatic injuries on the body.

**Internal examination:** FA and FP detached from dura mater, red leptomeningeal, apparent bilateral perithyroidal IH, cross section of the sternum - ossification nucleus, oval, violacein, 0.2-0.3 cm, cross section of ED femur - Beclard ossification nucleus of 1, 3 cm, pleural effusions - reddish brownish-red liquid, unresponsive lung, fluid in the small peritoneal cavity, generalized stasis, autolysis.

**Umbilical cord:** Length - 42 cm, thickness - 1.5 cm, shiny, purple, without ring on the implantation limit, no bleeding on the surface, section and though, clots observed in umbilical veins blood

**Placenta:** weight - 720 g, glossy fetal face with intensely congestive, turgescence blood vessels. In the lumen there were blood clots. Pocal, shows small bleeding areas and small yellowish-green areas, mature, lobular endometrial face with small bleeding zones and adherent blood clots, fibrous trabecular section, congestive vessels with intraluminal thrombi, macroscopic calcification, thin membranes, glossy, intensely congestive.

**Histopathological examination:** placenta - has acute focal inflammatory infiltrate, reduced in fetal membranes, vessels with arteriolar media hypertrophy, abundant fibrinoid, intervillus hemorrhage and deciduous hemorrhage, rare intervillos thombi, many syncytial nodes, congestive intensive vessels: no presence inflammatory infiltrate at the villous level; umbilical cord - with a highly congestive tri-vascular structure, without the presence of conjunctival organized thrombi, interstitial edema and marked autolysis of Warthon gelatin; there is no evidence of acute inflammatory infiltration of the umbilical cord, unresponsive lungs, undisturbed airspace; edema fluid associated with red blood cells can be seen at intra-alveolar level, numerous interstitial hemorrhages; immature-looking brain, intra-cerebral passive vascular congestion; diffuse edema, perivascular microhemorrhage, cord with a passive vascular congestion, fetal-shaped kidney and diffuse passive vascular congestion; interstitial hemorrhagic areas, marked autolyze changes. Pancreas and spleen - changes in autolysis and rotting marked by the deletion of characteristic cytorethycocytocya, liver - persistent rare portobilar spaces, deletion of hepatocyte cell boundaries, congestion of large interlobular vessels, changes in gouache, fragment of the umbilical cord insertion - thin epidermis detached from the surface of the dermis, without the presence of acute inflammatory cells, epicranium tegument fragment - numerous hair follicles, marked capillary congestion, advanced autolytic changes.

**DISCUSSION**

For the current case, it might be considered several details such as:

AHC – DZ non-insulin dependent mother, smoker, obese (72 kg at 8th week – 94 kg at the labor time); the pregnancy evolution: IA at the weeks 7, 10, 24, 30, LA slightly reduced for VG at 24th and 38th weeks of age;

7 days before the admission the glycemia was 126,02/mg/dL while the normal range is 60 - 110/mg/dL;

from the medical documentation cannot be figured out the fetal biometry;

the pregnancy evolution after admission – the admission occurred at 11.06 with MI, CUD non-systematized (MI)/systematized (IB); at 10.55 the uterine col with OE permeable at two fingers, MI (local examination), at 23.45 she is moved to the birthplace with the membranes broken by nature, until 06.00;

the newborn at the birth time: the weight 4660...
During the necropsy examination, the newborn was found dead, based on the lack of the bosom serosanguinous, CO without ring at the limit of implantation, P occupies: 1/3 of the thoracic cavity and have violet colour, of tough consistency, without crepitation, the same aspect, it is drained a small amount of dark blood, the hydrostatic docimasement the pulmonary fragments do not float to the surface of the water, the stomach contains a viscous fluid in the small amount of light grey colour, greenish intestines, viscous liquid light green dark in lumen. There have been signs of intrauterine death of the newborn such as red skin tears with detachment of the epidermis on the body surface, the bone of the cranial cap with spontaneous detachment from the hard tissue, pleural cavities and the peritoneal cavity contain a small quantity of red-brown liquid, autolyzed organs.

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CONCLUSION

The present case is of a newly born female, macromos, born dead. For the intrauterine death of the fetus it advocates lungs with an unresponsive appearance, the stomach containing a viscous liquid in a small amount of white ish colour, greenish-colored intestines, viscous liquid of greenish-blackish colour, umbilical cord without a demarcation ring at the implantation limit and lack of serous bosa. The macroscopic and microscopic examination of the necropsy advocates the possibility of the death of the fetus at least 24 hours before birth. For this, it advocates the following changes: skin detachment of the epidermis on the extended body surface, the bones of the skull with spontaneous detachment at the level of the hard tissue, pleural cavities and the peritoneal cavity contain a small amount of reddish-brown liquid, autolyzed organs. The intrauterine death of the fetus was most likely due to intrauterine hypoxia, without having any forensic evidence for the cause of hypoxia. Notice that there were no signs of violence on the body during the necropsy examination.

Conflict of interest. The authors declare that there is no conflict of interest.

References

10. Silver RM. Fetal Death, Obstetrics & Gynecology. 2007; 109(1).